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AMENDMENT AND RESPONSE TO OFFICE ACTION

HLA class I complex (or equivalent) on the surface of cells to be killed, wherein the HLA class I complex (or equivalent) type presenting the peptide in the cells to be killed is not present in the CTLs to be administered to the patient, and

the CTLs kill the presenting cells.

Please cancel claim 4.

- 5. (Three amended) A method according to Claim [4] 1 wherein the [polypeptide] antigen is a mutant polypeptide associated with the [diseased] cells to be killed.
- 6. (Three amended) A method according to Claim [4] 1 wherein the [polypeptide] antigen is present at an abnormally elevated amount in the [diseased] cells to be killed compared to [non-diseased] other cells.
- 7. (twice Amended) A method according to Claim 1 wherein the [disease is a] cells to be killed are cancer cells.
- 9. (twice Amended) A method according to Claim 1 wherein the [disease is caused by] cells to be killed have a chronic viral infection.
- 12. (twice Amended) A method according to Claim 1 wherein the [disease is] cells to be killed are associated with an abnormally elevated amount of a hormone.
- 13. (twice Amended) A method according to Claim 1 wherein the [disease is a bacterial disease caused by] cells to be killed have a chronic bacterial infection.
- 17. (twice Amended) A method according to Claim 14 wherein the cytotoxic T lymphocyte is selected from a library of CTL clones, the library comprising a plurality of CTL

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clones derived from individuals with differing HLA class I (or equivalent) molecule type and each CTL clone recognises the [diseased] cells to be killed.

18. (twice Amended) A method according to Claim 17 wherein each CTL clone recognizes at least part of the same molecule contained in or associated with the [diseased] cells to be killed.

Please cancel claims 25 and 26

27.(Three times amended) A method according to Claim 1 wherein the [molecule] antigen is selected from the group consisting of cyclin D1, cyclin E, mdm 2, EGF-R, erb-B2, erb-B3, FGF-R, insulin-like growth factor receptor, Met, myc, a p53, BCL-2, [mutant p53,] a polypeptide associated with the BCR/ABL translocation in CML and ALL, [mutant] a CSF-1 receptor, [mutant] an APC, [mutant] a RET, [mutant] an EGFR, a polypeptide associated with PML/RARA translocation in PML, a polypeptide associated with E2A-PBX1 translocation in pre B leukaemias and in childhood acute leukaemias, human papilloma virus proteins, Epstein-Barr virus proteins, HTLV-1 proteins, hepatitis B virus proteins, hepatitis C virus proteins, herpes-like virus proteins and HIV encoded proteins.

Please cancel claims 28 and 29.

## Remarks

## The Interview

Applicants and the undersigned greatly appreciate the opportunity to discuss this case with the examiner. The foregoing amendments to the claims are made in response to the